SEVERE PULMONARY HYPERTENSION WITH SIGNIFICANT "A" DIP ON PULMONIC VALVE ECHOCARDIOGRAM

Tulio A. Sulbaran, M.D., Efrain Garcia, M.D., Wayne E. Dear, M.D., Jorge Guttin, M.D., Leonard W. Pechacek, and Robert J. Hall, M.D.

ABSTRACT

A patient with severe pulmonary hypertension and no evidence of right ventricular failure who had a 4 mm "a" dip on the pulmonic valve echocardiogram is reported. Although other echocardiographic abnormalities suggesting pulmonary hypertension were recorded in our patient, the normal "a" dip of the pulmonic valve in the absence of right ventricular failure appears to be an exception to previously reported findings. We suggest motion of the entire pulmonary artery as an explanation for this phenomenon.

INTRODUCTION

Recent publications have emphasized the diagnostic value of echocardiography in studying patients with pulmonary hypertension.[1-5] Particular attention has been placed on the "a" dip which was absent or greatly diminished on the echocardiogram of all cases reported (provided no right heart failure was present) and has been considered reliable evidence in the diagnosis of pulmonary hypertension. A case is described which we believe to be the first report of a patient with documented severe pulmonary hypertension and no right heart failure, in whom we observed an "a" dip of 4 mm on echocardiographic study.

CASE REPORT

A 29-year-old woman was in good health until two years before admission to St. Luke's Episcopal Hospital on October 6, 1975. During the two years she had developed dyspnea upon exertion with increasing severity. She experienced no orthopnea or paroxysmal nocturnal dyspnea and denied chest pain, hemoptysis, cyanosis or syncopal episodes.

The patient appeared to be in good health and in no acute distress. The blood pressure was 100/60 mm Hg and the heart rate was 80 beats per minute. There was no cyanosis or clubbing, and jugular venous pressure was normal. The lungs were clear. The point of maximal impulse was in the

From the Division of Cardiology of St. Luke's Episcopal Hospital-Clayton Foundation for Research Laboratory, Houston, Texas.

Address for reprints: Efrain Garcia, M.D., St. Luke's Episcopal Hospital, P.O. Box 20269, Houston, Texas 77025.

fifth intercostal space at the midclavicular line, and a right ventricular heave was palpable. The first heart sound was normal, the second sound was persistently split and the pulmonic component was accentuated. There was a pulmonic ejection click and a faint systolic ejection murmur along the left sternal border. Results of electrocardiography showed regular sinus rhythm, a QRS mean axis in the frontal plane of +120 degrees, and right ventricular hypertrophy and strain. Cardiomegaly was evident on chest roentgenogram with main pulmonary artery segment prominence, but the pulmonary vascular markings were within normal limits. The remainder of laboratory studies, including SMA 12*, electrolytes, urinalysis, partial thromboplastin time, prothrombin time and coagulation time, were within normal limits. The hemoglobin was 15.9 gms%. Right and left cardiac catheterization was performed (Table I). The left side of the heart was

TABLE I. Cardiac Catheterization Data in Patient with Severe Pulmonary Hypertension and Significant "A" Dip on Pulmonic Valve Echocardiogram

Site	O_2 Saturation (%)	Pressure (mm Hg)
Superior vena cava	73.5	-
Inferior vena cava	74	-
Right atrium: High	76	-
Mid	75	$\underline{a}$ = 10; $\underline{v}$ = 5; $\overline{5}$
Low	73	-
Right ventricle	73	100/5-10
Pulmonary artery	76	100/42; $\overline{70}$
Left atrium	90.5	$\underline{a}$ = 5; $\underline{v}$ = 5; $\overline{2}$
Left ventricle	91	115/4-10
Aorta	91	115/70; $\overline{95}$

$\underline{a}$ = a wave; $\underline{v}$ = v wave

*SMA 12 = calcium, inorganic phosphatase, glucose, total protein, albumin, total bilirubin, alkaline phosphatase, lactic dehydrogenase, serum glutamic oxaloacetic transaminase, blood uric nitrogen, cholesterol, uric acid.

entered through the patent foramen ovale. There was no evidence of a left-to-right shunt demonstrated by a negative hydrogen curve. The cardiac index was 3.95 1/min/m^2. The pulmonary vascular resistance was 941 dynes/sec/cm^{-5}, and systemic vascular resistance was 1411 dynes/sec/cm^{-5}.

The findings of echocardiographic study included an enlarged right ventricle (3.9 cm), good left ventricular contractility, and normal septal motion. A decrease (40 mm/sec) was noted in the diastolic descent of the anterior leaflet of the mitral valve with normal motion of the posterior leaflet of the mitral valve (Fig. 1). There was a decrease (5 mm/sec) in

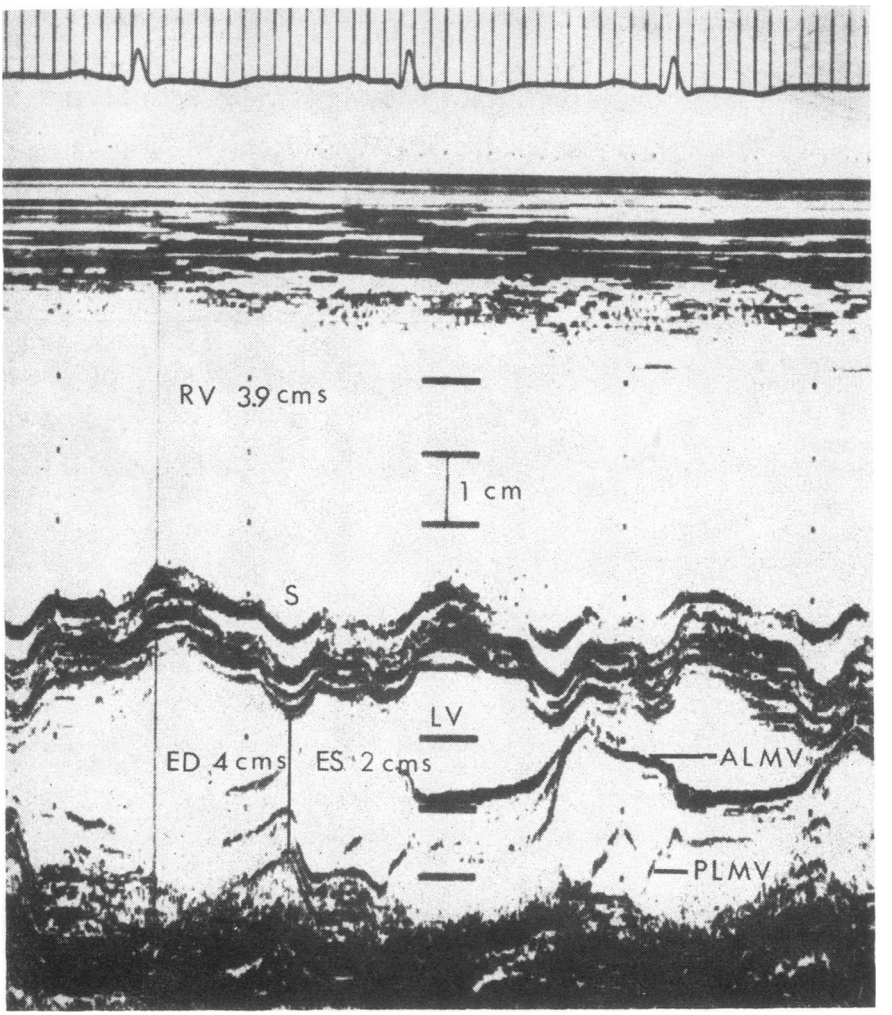

Fig. 1. Echocardiogram showing enlarged right ventricle (RV) with normal motion of non-thickened septum (S). LV, left ventricle; ED, end-diastolic diameter of the left ventricle; ES, end-systolic diameter of the left ventricle; ALMV, anterior leaflet of the mitral valve; and PLMV, posterior leaflet of the mitral valve.

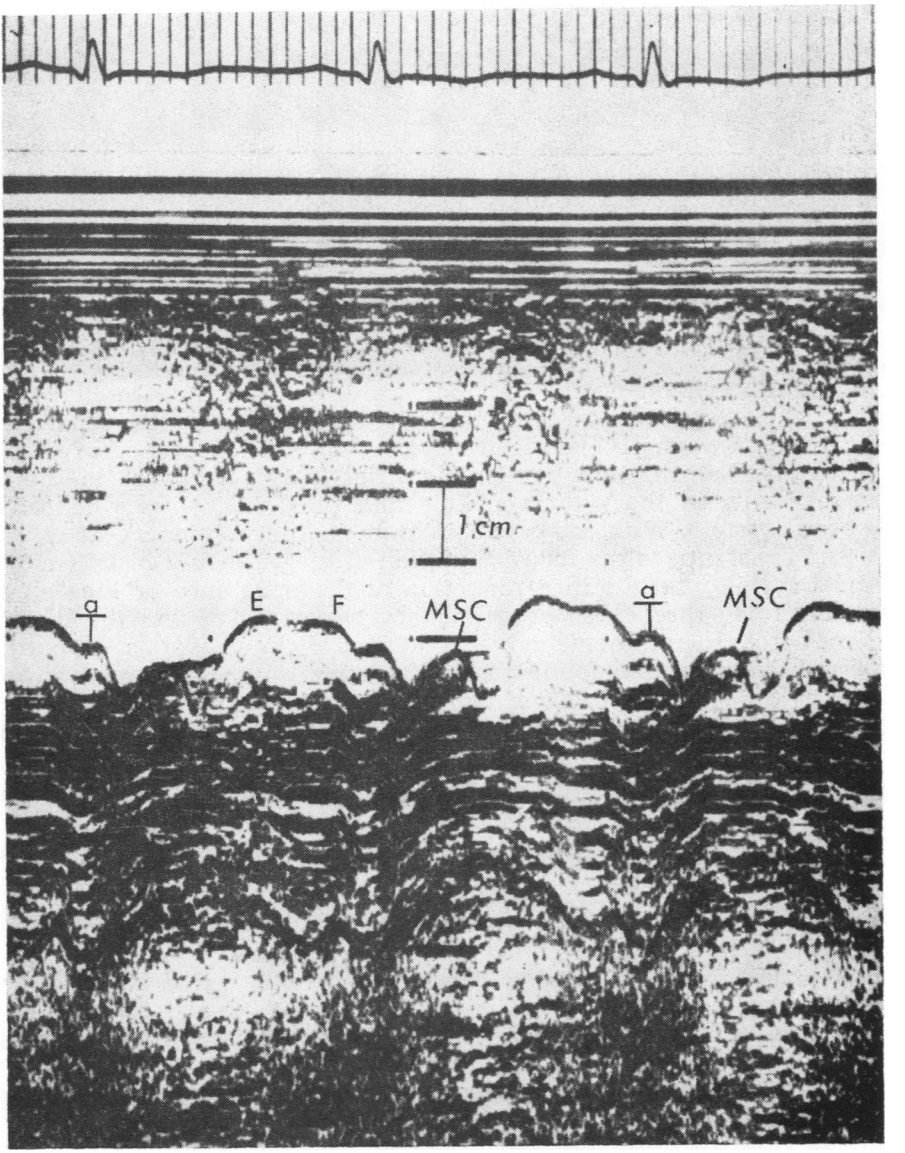

Fig. 2. Echocardiogram of the pulmonic valve showing prominent "a" dip (4 mm), decreased EF slope, and mid-systolic closure of the pulmonic valve (MSC).

the EF slope of the pulmonic valve, midsystolic closure of the pulmonic valve, a preejection period (corrected by rate) of 95 msec and an "a" dip of 4 mm with no respiratory variation (Fig. 2). The left atrium, aortic root, and aortic valve were normal.

DISCUSSION

Several authors have described features of pulmonary hypertension evident on echocardiographic study. Enlargement of the right ventricle and reduced diastolic descent of the anterior leaflet of the mitral valve, with normal motion of the posterior leaflet were consistent findings in the nine cases reported by Goodman, et al.[1] Others,[2,3] have described a flattened (decreased) diastolic slope, rapid opening slope in systole, prolonged preejection period, midsystolic closure or notching of the systolic segment, and diminished or absent "a" dip on the pulmonic valve echocardiogram.

The "a" dip evident on the pulmonic valve echogram represents movement of the cusps toward the open position and has been reported to follow the "p" wave on the electrocardiogram. Disappearance of the "a" dip with atrial fibrillation further identifies it as a response to the low pressure events in the right ventricle and pulmonary artery associated with atrial systole. With increased pressure in the pulmonary artery, the increased resistance to the forward motion of the pulmonic cusps (induced by atrial systole) results in a reduction or disappearance of the "a" dip, depending upon the degree of pulmonary hypertension and the diastolic gradient across the pulmonic valve. Nanda, et al[3] in a report of 25 patients in normal sinus rhythm found that when mean pulmonary pressure was 20-40 mm Hg, a small "a" dip of less than 2 mm was present, while more severe hypertension [$\geq$ 40 mm Hg (mean)] was characterized by the absence of the "a" dip except in patients with severe right ventricular failure. When the diastolic gradient across the pulmonary valve was over 20 mm Hg, no deflection attributable to atrial contraction was present. In a report of 24 patients with pulmonary hypertension and sinus rhythm, Weyman and associates[2] found no patient to have an "a" wave of over 2 mm.

Our patient was remarkable in that she displayed a large "a" dip on echocardiography in the presence of severe pulmonary hypertension, no right ventricular failure, and a diastolic pulmonic valve gradient well above 20 mm Hg. The echocardiogram displayed several features compatible with pulmonary hypertension. The pulmonic valve had a decreased E-F slope, midsystolic closure, and lack of respiratory variation of the "a" dip. These findings, combined with right ventricular enlargement, normal septal motion, and a decreased E-F slope of the anterior leaflet of the mitral valve with normal posterior leaflet motion strongly suggest the correct diagnosis even in the presence of a normal "a" dip. The reason for this finding is obscure and indicates other unrecognized factors in the genesis of the negative deflection "a" dip on echocardiography. A possible explanation is that the "a" dip, although originated by atrial contraction, reflects the motion of the entire pulmonary artery and not the pulmonic valve alone. Unfortunately, the pulmonary artery cannot be visualized with M Mode echocardiography in order to prove this hypothesis. How-

ever, cross sectional echocardiography and/or angiography may aid in establishing such a correlation.

In summary, although the "a" dip is diminished or absent in most patients with pulmonary hypertension, a large "a" dip can occur in the presence of a pulmonary artery-to-right ventricular diastolic gradient greater than 20 mm Hg and a pulmonary artery mean pressure greater than 40 mm Hg.

REFERENCES

1. Goodman D, Harrison D, Popp R: Echocardiographic features of primary pulmonary hypertension. Am J Cardiol 33:438-443, 1974
2. Weyman A, Dillon J, Feigenbaum H, Chang S: Echocardiographic patterns of pulmonic valve motion with pulmonary hypertension. Circulation 50:905-910, 1974
3. Nanda N, Gramiak R, Robinson T, Shah P: Echocardiographic evaluation of pulmonary hypertension. Circulation 50:575-581, 1974
4. Sakamoto T, Matsuhisa M, Hayashi T, Ichiyasu H: Echocardiogram of the pulmonary valve. Jap Heart J 7:360-373, 1974
5. Weyman A, Dillon J, Feigenbaum H, Chang S: Echocardiographic patterns of pulmonary valve motion in valvular pulmonary stenosis. Am J Cardiol 34:644-651, 1974